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ETIOLOGY AND TREATMENT OF AURICULAR FIBRILLATION AND AURICULAR FLUTTER

I. Auricular Fibrillation

ETIOLOGY. There is no specific etiological factor known in auricular fibrillation, but it is recognized that this arrhythmia is most commonly observed in association with certain clinical conditions. Evidence of rheumatic heart disease is found in from 40 to 65% of the cases; most of these fall in the younger age group (3rd and 4th decades). Non-valvular heart disease is found in from 30 to 40%; these usually fall in the older age group (6th and 7th decades). Hyperthyroidism is noted in 10 to 15%, and other toxic states (infections, drugs, etc.) in 2 to 5%. The association of auricular fibrillation with syphilitic heart disease is very uncommon. In 2958 cases of this arrhythmia collected from the literature the incidence of syphilis was found to be approximately 1.5% (Evans). Four to nine per cent of the cases of auricular fibrillation disclose no evidence of any other disease. The arrhythmia is infrequent in persons under twenty years of age, and very rare in children under twelve years. Sex appears to have no significant influence on the occurrence of this condition.

VARIETIES OF FIBRILLATION. Auricular fibrillation is classified as (1) paroxysmal (or transient), and (2) permanent (or persistent or established). The distinction is purely a clinical one and is determined by the duration of the attack. In more than 90% of the cases of paroxysmal auricular fibrillation the attacks cease spontaneously in from a few minutes to one week. If, therefore, an attack lasts longer than seven days it is more or less arbitrarily regarded as belonging to the permanent variety (Friedlander and Levine, Parkinson and Campbell). The onset of persistent auricular fibrillation in many instances is preceded by transient attacks over a variable period of time.

REMEDIES. The chief remedies employed in auricular fibrillation are digitalis and quinidine. Although in many respects the two drugs produce on the cardiac function opposite pharmacological effects, in clinical fibrillation their actions are complementary. Adequate digitalization prior to the administration of quinidine "probably increases the chances of success and diminishes the danger of accidents. It prevents the ventricular tachycardia that sometimes occurs in quinidine therapy. Conversely, quinidine counteracts the 'toxic irregularities' of digitalis" (Sollman).

CONDITIONS AFFECTING THE CHOICE OF THERAPY. The principal conditions which affect the choice of therapy in the management of auricular fibrillation are etiology, variety of fibrillation, apical heart rate, and congestive failure.

The significant factors pertaining to etiology which are taken into consideration in connection with treatment are the presence of organic heart disease, the absence of evidence of heart disease apart from the arrhythmia, and associated hyperthyroidism.

AURICULAR FIBRILLATION WITH ORGANIC HEART DISEASE. In advanced organic heart disease with auricular fibrillation of long standing, the best results are usually obtained by adequate digitalization, sufficient to maintain a reasonably slow rate (70 to 80 per minute) and to reduce the pulse deficit to a minimum. In such instances it is probably undesirable to attempt to abolish the arrhythmia by means of quinidine, for the normal rhythm thus established is not likely to be permanent and the benefits derived therefrom are not sufficient to compensate for the risks involved in the use of the drug. It is generally recognized that it is in this type of case that the more dangerous untoward effects of quinidine (embolism, collapse and sudden death) are most apt to manifest themselves. On the contrary, occasional cases of organic heart disease with congestive failure appear to derive some advantage from auricular fibrillation since the heart rate may be slowed to more efficient levels with digitalis and the response to that drug is often better than when the rhythm is normal. It has been noted also that auricular fibrillation reduces very greatly the possibility of the development of subacute bacterial endocarditis, and sometimes lessens the severity of the pain in angina pectoris.

Notwithstanding these apparent advantages of auricular fibrillation, there are many obvious disadvantages in the continuation of the abnormal rhythm. Perhaps the most important of these is the relationship of the arrhythmia to the occurrence of embolic accidents. Although it is held by some authorities (Lewis, Cookson) that, except under quinidine therapy, embolism is no more common in cases with auricular fibrillation than in similar cases with normal rhythm, it is generally accepted that this arrhythmia predisposes to the formation of auricular thrombi.* It is also fair to assume that if the auricular fibrillation is terminated shortly after its inception the chances for the formation of such thrombi are greatly reduced. The conclusion therefore seems justified that whenever possible without too great risk, the attempt should be made to establish normal rhythm. In auricular fibrillation of short duration (less than six months) occurring in cases where the organic heart changes are not too far advanced, the dangers attending the use of quinidine are relatively less serious. Hence, in such cases quinidine preceded by digitalization may be used with probable benefit and comparative safety.

The use of quinidine is also worthy of trial in the more advanced cases of chronic heart disease with severe decompensation which fail to respond to digitalis or any other form of treatment. Cases of this type have been reported (Van Nuys; Kohn and Levine) in which quinidine used as a last resort established normal rhythm and restored compensation.

*"Increasing years" is another important factor favoring auricular mural thrombosis (Kohn and Levine).

AURICULAR FIBRILLATION WITH NO OTHER EVIDENCE OF HEART DISEASE. In cases of auricular fibrillation without other evidence of heart disease, quinidine is the most effective therapeutic agent and is the therapy of choice. As already pointed out, the chances of success are increased by preliminary digitalization. After such preparation, a test dose of 0.2 gram of quinidine is administered. In some instances this single "test" dose is sufficient to restore a normal rhythm. If, however, the arrhythmia continues and no untoward effects from the test dose develop in six to twelve hours, the administration of the drug is resumed in doses of 0.3 to 0.4 gram at four to six hour intervals until normal rhythm is restored or until the appearance of toxic symptoms necessitates the discontinuance of the drug. After the restoration of the normal rhythm, a daily maintenance dose of 0.2 to 0.4 gram or more may be required to prevent recurrence of the arrhythmia. Often the rhythm will remain regular even if the drug is stopped abruptly after the cessation of fibrillation. In this type of case quinidine is almost invariably successful in restoring normal rhythm, and the patient often remains well indefinitely without further treatment.

AURICULAR FIBRILLATION WITH THYROTOXICOSIS. In thyrotoxicosis, auricular fibrillation may require no special treatment, since the adequate management of the primary cause often will result in a spontaneous cessation of the arrhythmia. If, in the absence of organic heart disease, such desired results fail to develop within one to three weeks after thyroidectomy, quinidine therapy will usually restore normal rhythm. A resumption of fibrillation after such restoration of regular rhythm usually signifies either that an insufficient amount of the gland was removed, or that some other additional cause was responsible for the arrhythmia.

In hyperthyroidism with normal rhythm, the important indication is the control of the thyrotoxicosis. The early use of iodine, glucose and surgery will tend to prevent the appearance of auricular fibrillation. It is questionable whether quinidine should be used prophylactically in such instances, but in small doses it will certainly do no harm. Statistics bearing on this point are not now available.

CHOICE OF THERAPY WITH REFERENCE TO TYPE OF AURICULAR FIBRILLATION. The paroxysmal type of auricular fibrillation often requires no special treatment. In about 80% of the cases the paroxysms cease spontaneously in less than two days; in many the attacks last but a few minutes to a few hours; and only in very rare instances does a typical attack continue longer than one week (Parkinson and Campbell). In paroxysms of relatively long duration treatment is indicated in accordance with the general principles already outlined. Quinidine in doses of 0.1 to 0.4 gram twice or three times daily may also be used prophylactically in cases subject to frequently recurring attacks.

EFFECT OF APICAL RATE UPON CHOICE OF THERAPY. Auricular fibrillation associated with a slow apical rate not the result of treatment is quite rare and usually signifies the presence of organic heart block. In such cases no special therapy is required apart from the measures indicated by the underlying cardiac condition. However, in the very rare instances where auricular fibrillation and slow rate are associated with an otherwise normal heart, an attempt should be made to abolish the arrhythmia with quinidine.

INFLUENCE OF CONGESTIVE FAILURE WITH NO OTHER EVIDENCE OF HEART DISEASE UPON CHOICE OF THERAPY. Congestive failure is generally regarded as an indication of organic heart disease. While this is undoubtedly true in the great majority of cases, there is evidence available that auricular fibrillation apart from any other disease of the heart may induce such failure. It is therefore important in every case of congestive failure with auricular fibrillation to determine the nature of the underlying cardiac disease. If no organic heart disease can be demonstrated it is sometimes possible to bring about complete and lasting recovery by digitalization and quinidine therapy. A case of this type was reported by Parkinson and Campbell. The arrhythmia was successfully terminated by means of quinidine and subsequent to recovery the patient was observed for a period of five years during which time he continued in normal health without further treatment. The author has under observation a similar case. It is now more than one and one-half years since all treatment was discontinued and the patient remains in perfect health.

II. Auricular Flutter

Auricular flutter is very closely related to auricular fibrillation. The mechanism of its production is essentially the same except that in flutter the circus movement is more regular and much slower in rate. It occurs under the same circumstances as auricular fibrillation but is encountered much less frequently, the ratio being about one to twenty (Parkinson and Bedford). The same remedies are employed in both arrhythmias. However, in auricular flutter quinidine is relatively less important since digitalis alone is usually effective in restoring normal rhythm. The initial effect of digitalis is to render the rate slower and more irregular. Upon complete digitalization the rhythm is often converted into auricular fibrillation. After digitalis is discontinued normal rhythm is usually resumed. If such result is not obtained within a few days, quinidine may then be employed.

III. Treatment of Quinidine Intoxication

The most frequent early symptom of quinidine intoxication is tinnitus and others less commonly seen are nausea, vomiting, epigastric distress and diarrhea. More rarely there may be headache, palpitation, fear, mental depression, flushing, urticaria, sweating, syncope, and tachycardia. Ventricular ectopic beats may precede ventricular tachycardia and ventricular fibrillation. The occurrence of embolic phenomena is more common under quinidine therapy than under digitalis alone. Sudden death other than of embolic origin is also occasionally observed, probably due to ventricular fibrillation or to respiratory paralysis (Kohn and Levine). If such an emergency is anticipated, life may sometimes be saved by artificial respiration and the intravenous or intracardiac administration of large doses (15 grains) of caffeine sodio-benzoate.

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Editorial Addendum. Whereas the clinical diagnosis of auricular fibrillation or auricular flutter is generally confirmed by electrocardiogram, certain comment should be made on clinical signs. Auricular fibrillation is, as is commonly known, diagnosed by the absolute irregularity of the heart, but when the ventricular rate is rapid, and especially when the arrhythmia is paroxysmal, it may be confused with paroxysmal ventricular tachycardia. When the ventricular rate is slow it may be confused with multiple ectopic beats, auricular, ventricular, or both.

Auricular flutter should be suspected if a rapid regular rate, especially from 100-120 beats per minute, does not vary with activity but slows on pressure over the carotid sinus. Flutter waves are occasionally observed in the jugular venous pulse. The heart's rate may become slow and the rhythm irregular after digitalis administration.